

medicine? The answer: focused, gender-specific, cardiovascular clinical research.

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REFERENCE

1. Anand SS, Xie CC, Mehta S, et al. Differences in the management and prognosis of women and men who suffer from acute coronary syndromes. *J Am Coll Cardiol* 2005;46:1845–51.

REPLY

In the gender subgroup analysis of the CURE (Clopidogrel in Unstable Angina to Prevent Recurrent Events) trial data, we observed that fewer women across all Thrombolysis In Myocardial Infarction (TIMI) risk strata underwent coronary angiography compared to men (1). However, among women who did have significant coronary artery disease (CAD), an equal proportion went on to have coronary revascularization compared to men. We also noted, however, that women in the high-risk acute coronary syndromes (ACS) group were significantly more likely to have refractory angina or be readmitted to a hospital with recurrent angina as compared to men. Dr. Kessler is correct that the differences in revascularization rates could not account for the difference in refractory ischemia/rehospitalization rates we observed. However, if women were equally as likely to have coronary angiograms as men, then more women with significant CAD may have been identified. Subsequent revascularization in this high-risk group may have equalized the rates of refractory ischemia between women and men.

However, we recognize that our association represents a subgroup analysis, which at best can raise a hypothesis but cannot confirm whether it is true. Like Dr. Kessler, we also advocate sex- and gender-based research in cardiovascular disease using sufficient sample sizes and methodology to be confirmatory.

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Troponin Trumps Common Sense

The state-of-the-art review of biomarkers in acute cardiac disease by Drs. Jaffe, Babuin, and Apple is thoughtful and timely (1). My concern, shared by many cardiologists, is that troponin testing has gotten out of hand. Most cardiologists have been asked to see a patient (often urgently) found to have a mildly elevated troponin when the test was ordered reflexively—regardless of the patient's presenting complaints or past history. Cardiologists on hospital services are tripping over troponin every day. The most challenging situation is when a patient presents with a serious (even life-threatening) noncardiac condition, and one or more doctors involved in their care gets distracted by an incidental mild troponin elevation. Occasionally, this leads to a sudden obsession over one test result, a phenomenon I call “troponin trumps common sense.”

Several problems are associated with the uninformed use of this sensitive assay. The authors present information about the *appropriate* use of troponin testing that should be actively diffused into practice. They write, “Because of the sensitivity of cTn [cardiac troponin], elevations are common in patients with a large number of acute and chronic cardiovascular diseases. It is up to the clinician to decide whether the presentation is one of acute ischemia.” (1) Table 2 (1) lists about two dozen situations where “elevations of troponin in the absence of overt ischemic heart disease” occur. Admittedly, most patients presenting with an acute coronary syndrome (ACS) also have one or more of the conditions listed. This is where clinical judgment counts. The patients I am describing *do not* present with chest pain, dyspnea, or other symptoms and signs, or an electrocardiogram suggesting an acute cardiovascular problem.

Consider the cost of all the unnecessary stress tests ordered, coronary angiograms performed, and anti-platelet agents prescribed for mild troponin elevations when the clinical situation makes an acute cardiovascular problem very unlikely. The casual use of the phrase non-ST-segment elevation myocardial infarction (NSTEMI) when the mild troponin elevation is not, in fact, due to atherosclerotic coronary artery disease, creates its own legacy. Think twice before attaching the NSTEMI label to a patient with a mild troponin elevation much more likely to be due to one or more of the conditions outlined by the authors in Table 2 (1).

It is useful to draw an analogy between mild troponin elevations and *nonspecific* ST-T changes on an electrocardiogram. I suggest using the descriptive phrase “nonspecific mild troponin elevation” if there is no compelling evidence to support a diagnosis of an ACS and in patients with chronic cardiovascular disease or noncardiac diseases. Doctors do not feel compelled to request an urgent cardiology consult on every patient with nonspecific ST-T changes on an electrocardiogram in the absence of any cardiac symptoms or history of cardiac disease. Rather than allowing troponin to trump common sense, we should inject more common sense into the process of ordering a troponin level in the first place.